Pain sensitivity, exercise and stoicism

Malvin N Janal PhD

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INTRODUCTION

Habitual recreational runners engage in strenuous physical activity undeterred by fatigue, pain, or adverse environmental conditions. Some continue to run despite stress fractures[1] or the onset of myocardial infarction[2]. Even without injuries, however, regular training produces regular discomfort. These observations raise two questions: are athletes generally less sensitive to pain than normally active persons, and does exercise reduce pain sensitivity?

As the presence or magnitude of allegedly noxious natural stimuli cannot be controlled, laboratory pain tests are employed. By presenting uniform, calibrated noxious stimuli to athletes and controls, or to athletes under different experimental conditions, one can reasonably study differences in pain responsivity. This paper reviews evidence of differences in pain responsivity between habitual exercisers and normally active people and of changes in pain responsivity during exercise. It further discussses the notion of 'stoicism' with regard to these findings.

Various laboratory pain tests have been utilized to document the effects of exercise on pain behaviour. To facilitate later discussion, these stimuli will be described briefly. The tourniquet ischaemia pain test[3] usually involves inflating a pressure cuff above systolic pressure on the upper arm, after which the subject exercises the hand for several minutes. Sensation and pain reports are elicited at regular intervals, and the subject is encouraged to tolerate the stimulus for as long as 10 or 15 min. Cold pain is produced by the cold pressor test, in which the subject immerses his hand in an ice-water slurry for as long as 3 to 5 min. During this immersion, sensation and pain reports are elicited at regular intervals, and the time to withdrawal is noted. Heat pain is often delivered by a contact thermode[4], but also with focused light[5] and lasers[6], either in a continuous ramp or as temporally discrete stimuli. The subject either rates each discrete stimulus on a rating scale, or indicates when particular levels of sensation or pain have been achieved along the ramp. Electrical stimuli are delivered to the skin, usually of the fingers or forearm[7], but also to the teeth[8–10]. The stimulation and response protocols are analogous to those described for thermal stimulation. Pressure pain is usually produced by placing the subject's finger under the dulled edge of a lucite block; different forces press the edge against the dorsal surface of the finger, and the subject reports when particular levels of sensation or pain have been achieved[11].

Responses are then summarized by various psychophysical measures. Most commonly, the pain threshold is taken as the mean stimulus intensity evoking the minimal report of pain. Tolerance is taken as the mean maximal intensity endured on the trials. Another model, sensory decision theory (SDT)[12], provides two indices of perceptual performance. Discriminability indexes the accuracy in separating higher from lower intensity stimuli, and response bias indexes the most probable report category. Since most of these indices can vary independently of one another, the use of SDT separates measures of average report level from sensitivity to differences in level. Analgesics such as local nerve blocks or IV morphine[5] reduce both the number of pain reports and discriminability, while placebos only reduce the number of pain reports[13].

DOES PAIN PERCEPTION DIFFER IN RUNNERS AND NON-RUNNERS?

Do people who exercise regularly report less pain than those who do not? When both groups are tested without recent exercise, regular athletes showed similar thresholds for noxious heat or ischaemic stimulation as others, but their threshold for noxious cold was significantly higher than that of controls[14–16]. While this last finding supports the hypothesis of insensitivity, this singular increase can be more easily explained as a by-product of the resetting of the thermal set-point with chronic exercise[16]. Further, and contrary to the hypothesis, runners discriminated among noxious thermal stimuli significantly better than controls[16]. Thus, these data did not generally support the hypothesis of diminished pain thresholds in habitual runners.

On the other hand, these studies did show that runners were more tolerant of noxious ischaemic stimulation and noxious pressure, but not noxious cold, than normally-active individuals. Thus, regular athletes show greater pain tolerance than others, but similar pain thresholds. As only three studies have documented the athlete/control difference, these findings require replication and extension. If verified, many interesting questions could be posed regarding mechanisms of

Department of Biopsychology, New York State Psychiatric Institute, New York, USA

Correspondence to: Unit 50, 722 West 168th Street, New York, NY 10032, USA

action. For example, is increased tolerance genetic, or does chronic training change pain-reporting behaviour?

EXERCISE ANALGESIA STUDIES IN MAN

The second question concerns exercise-induced analgesia (more precisely, hypalgesia) during or after a period of exercise. Exercise analgesia is often taken as an example of stress-induced analgesia, where brief exercise is the stressor. A large literature[17] has demonstrated analgesia following many types of stress.

Reviewed below are studies which sought to document exercise analgesia as well as isolate factors which influence its occurrence and strength. Table 1 lists these studies, indicating the pain test used, the number of subjects tested, the duration of exercise, exercise intensity, and whether exercise was followed by evidence of opioid analgesia (assessed by administration of naloxone, a specific opioid antagonist).

Effects of different exercise durations

Results do not point to a consistent effect of exercise duration. Shorter duration exercise (less than 12 min) was followed by: increased pressure-pain thresholds after a 1-mile run[18]; increased dental and electrocutaneous thresholds after approximately 9–12 min of treadmill exercise[29]; and increased dental, thermal and electrocutaneous pain thresholds after as little as 8 min of bicycle ergometer exercise[9,10,19,20]. On the other hand, two studies (Janal, Glusman, Kuhl, Clark, in preparation)[22] failed to find analgesia after 9–15 min of increasingly faster and steeper treadmill exercise.

Studies using longer periods of exercise (20–40 min) have shown: increased ischaemic pain thresholds after a 40-min run[22,23]; reduced discriminability of painful heat[23,24]; increased dental pain thresholds[25]; increased nociceptive flexion reflex thresholds; and increased tolerance of the cold pressor test. On the other hand, two studies[23,28] failed to find analgesia on ischaemic pain, pressure pain, or cold pressor tests following a 40-min run.

Studies evaluating pain thresholds after varying durations of exercise[9,10,19,20] found that at least 8 min of exercise is necessary for an increase in dental pain thresholds; however, continuing exercise led to further increases in threshold, up to their exercise limit of 24 min. These studies suggest that longer durations produce greater reductions in pain reports. One study (Janal *et al.*, in preparation) tested pain perception in 12 regular runners under three exercise

Table 1 **Studies of exercise-analgesia in man, listed chronologically**

Author	*Test*	*N*	*Ex Time*	*Ex Intensity*	*Outcome*
Black *et al.* 1979 (Ref 22)	I	1	40	5 km	N-S analgesia
Haier *et al.* 1981 (Ref 18)	P	15	< 10	1 m	N-S analgesia
Janal *et al.* 1984 (Ref 23)	C, I, H	12	40	85% $VO_{2\,max}$	N-I thermal and N-S ischaemic analgesia; cold unaffected
Pertovaara *et al.* 1984 (Ref 19)	D, E	6	8 min × 3	50–200 W	Threshold increased with load of 50 W after 8 min
Kemppainen *et al.* 1985 (Ref 9)	D	7	8 min × 3	100–250 W	Threshold increased with work load of 100 W after 8 min
Kemppainen *et al.* 1990 (Ref 20)	D		8 min × 3	100–200 W	Threshold increased with intensity after 8 min
Olausson *et al.* 1986 (Ref 25)	D	11	20 min	HR=150	N-I threshold increase
Droste *et al.* 1988 (Ref 21)	E, I	17	9 min	Bruce	No analgesic effects
Droste *et al.* 1991 (Ref 8)	D, E	10	> 10 min	Bruce	N-S analgesia only at maximal work load
Vogel 1991 (Ref 28)	I, P	28	40 min	10 km	No analgesic effects
Guieu *et al.* 1992 (Ref 26)	E	6	20 min	200 W	Increased RIII flexion reflex threshold
Fuller and Robsinon 1993 (Ref 24)	H	22	40 min	10 km	d′ reduced for hot but not painful thermal stimuli
Padawer and Levine 1993 (Ref 27)	C	75	20 min	50/70% HR_{max}	Decreased ratings of cold pain
Janal *et al.* (unpublished)	C, I, H	60	9–15 min	Bruce	No analgesic effects
Janal *et al.* (unpublished)	C, I, H	12	40 min	80% HR_{max}	N-I decrease in thermal d′, N-I increase in ischaemic threshold

Ex=Exercise; I=ischaemic pain test; N-S=naloxone-sensitive; P=pressure pain tests; C=cold pressor test; H=heat pain test; N-I=naloxone-insensitive; D=dental pain test; E=electrical pain test; HR=heart rate; d′=signal detection theory parameter of sensory sensitivity; Bruce=standard exercise protocol for evaluating cardiac function (see Ref 19)

conditions: 15 min of treadmill exercise[29]; a 40-min treadmill run; and 40–50 min outdoor run. Exercise analgesia was apparent only after the outdoor run; ischaemic pain thresholds were increased and the discriminability of noxious thermal stimuli was decreased. These data suggest that between 15 and 50 min of exercise are needed to produce post-run analgesia.

The literature does not contain a study which varies exercise duration and intensity in a systematic way. However, Pertovaara *et al.*[19] and Kemppainen *et al.*[9,10,20] report analgesia following 8 min of exercise, irrespective of whether exercise intensity was 50 W or 100 W. Separating the effects of exercise duration from those of intensity deserves further attention.

In summary, shorter durations (at least 8 min) appear as likely as longer durations to produce exercise-analgesia, although greater increases in pain threshold may follow longer periods of exercise.

Stimulus modality

The success or failure of different studies to find exercise-analgesia may be related to differences in stimulus modality. Only one of four studies employing the cold pressor test found post-exercise analgesia. Therefore this test should probably not be employed in such studies. Three studies employing ischaemic pain found post-exercise analgesia, while three did not. Thus, this test is also inconsistent. Heat pain analgesia was found in four of five studies, and electrocutaneous pain proved sensitive to exercise in two of three studies. Finally, all four studies employing electrical stimulation of the teeth found a post-exercise analgesia. Dental stimulation thus appears to be the most reliable indicator of exercise analgesia effects.

Are effects confined to trained individuals

Earlier studies[18,23] employed only people who exercised regularly, leaving open the possibility that exercise analgesia was specific to them. Later studies[8–10,19,20], however, have demonstrated that regular exercise is not necessary, since analgesia was demonstrable in normally active people. Studies comparing exercise analgesia in athletes and normally-active controls would be of interest.

Opioid sensitivity

To test whether exercise analgesia is mediated by endogenous opioid mechanisms, many studies have challenged exercise-analgesia effects with the opioid antagonist naloxone. Most of these[8,18,22,23] have supported the hypothesis that exercise analgesia is mediated by an opioid mechanism, since naloxone reversed post-exercise analgesic effects on tests of ischaemic, pressure and dental pain. However, not all studies support the opioid hypothesis. Naloxone did not reverse post-run increases in dental pain thresholds[25], or reductions in thermal pain discriminability[23]. These studies suggest that exercise-analgesia may be mediated by both opioid and non-opioid mechanisms, and may interact as well with the particular test used to assess analgesia.

Pain system specificity

Does exercise produce analgesia specifically, or does it result in a general reduction in sensory perception? Studies have evaluated this question by assessing the perception of non-painful cutaneous stimuli, and by assessing other sensory modalities. Janal *et al.*[23] evaluated the effects of exercise on innocuous as well as noxious intensities of heat and ischaemia. Exercise diminished the perception of painful but not non-painful levels of heat, and painful as well as nearly painful levels of ischaemia. Janal *et al.* (in preparation) later evaluated the effects of exercise on an auditory SDT task. Whereas the post-run discriminability of noxious thermal stimuli was reduced, that of auditory stimuli was unchanged. Further, Droste *et al.*[8] showed a post-run reduction in magnitude estimates of electrocutaneous intensities that varied between 2.4 and 3.0 times the pain threshold intensity, but not for stimuli only 0.8 or 1.6 times that intensity. Post-run analgesia has been shown for threshold intensities of ischaemic and thermal pain, but not for higher intensities[23,24]. These studies are consistent with the hypothesis that perceptual alterations following exercise are specific to the pain system. Furthermore, exercise appears to influence the pain threshold more reliably than pain tolerance.

Experimental design issues

Most studies have employed within-subject designs, comparing pain sensitivity before and after exercise in the same individuals. As they lack a 'no exercise' control condition, thresholds may increase on re-test irrespective of the intervening condition. Padawer and Levine[27], who pointed out this problem, showed reduced ratings on the cold pressor test in groups who performed either 20 min of bicycle exercise or 20 min of (non-stressful?) painting with water colours. On the other hand, Fuller and Robinson[24] tested the same hypothesis, and showed analgesia after exercise but not after a 'no-treatment' control period. While the suggestion of using a no-treatment control is valid, between-subjects designs[27] require more subjects for an experiment, since they fail to capitalize on within-person consistencies in pain report. More efficiently, within-subject designs can counterbalance the order of exercise and control conditions, and use fewer subjects[24]. In addition to

'no-treatment' controls, we would also suggest that future studies pre-train subjects on the pain tests, so as to achieve a stable baseline before instituting either control or exercise manipulations.

Mechanisms

While general mechanisms of stress-induced anti-nociception may be invoked to explain exercise analgesia, for example the descending inhibitory pathways of the spinal cord dorsolateral funiculus[30], there have been few attempts to specify exactly how exercise interacts with those systems. Thoren *et al.*[31] presented a credible model of exercise-specific analgesia. The key element in their model involves afferent activity from A-delta fibres located in large muscle groups. Electrical stimulation of such fibres in animals has been shown to increase central opioid activity and to produce naloxone-reversible analgesia. Exercise of these large muscle groups has also been shown to produce activity in these afferent fibres. Thoren *et al.*[31] hypothesize that exercise also increases central opioid activity and reduces pain sensitivity. Even if correct, this model would not explain non-opioid anti-nociceptive effects.

STOICISM

How do these findings relate to the notion of stoicism? Since stoicism is not a well-defined construct, one point-of-view will be developed. First, we posit that stoics should *feel* as much pain as others, but *express* less. It is helpful here to distinguish, as Loeser[32] and Fordyce[33] do, between nociception (neural activity), pain sensation (sensory response), suffering (emotional response) and pain behaviour (illness and coping behaviours). Less pain is reported after a nerve block because nociception is impaired. Nociception is intact in stoics, but less pain is (honestly) reported because the sensory response is reduced, and/or suffering and pain behaviours are inhibited. Second, stoics would be expected to minimize the expression of all feelings, not just pain, and to do so consistently across time and circumstance. As stoicism is indicated by converging evidence about the lesser expression of pain and other feelings, it can be an appropriate explanation of fewer pain reports only when such a pattern of stoical behaviours has been demonstrated. Glusman *et al.*[34] successfully used this strategy to evaluate denial, a concept which also depends on converging evidence, in patients with silent myocardial ischaemia, measuring responses to several noxious laboratory stimuli as well as to questions about mood, anxiety, and coping style.

Stoicism should be distinguished from other traits (either innate or learned) which appear stoical, such as perseverance, which can influence pain behaviour but are not directly related to pain. To illustrate let us suppose that two athletes of similar talent and training are preparing for competition. One acknowledged discomfort during a long run, but did not seek to escape the situation. The other refused to acknowledge any discomfort. While both runners can be said to persevere, only the second would be considered stoical. While it may be difficult to distinguish the contribution of perseverance and stoicism to the report of any particular stimulus, this distinction must nevertheless be made.

One important consequence of the assumption that stoicism reflects a consistent response style is that situational reductions in pain report do not indicate stoicism. For example, compromising the neural substrate (e.g. by local nerve block), activating endogenous anti-nociceptive systems, (e.g. following an acute bout of exercise), or manipulating cognitive factors (e.g. with placebo instructions) would not constitute instances of stoicism. Thus, high thresholds during and after exercise are not evidence of stoicism. On the other hand, the greater pain tolerance shown by individuals who exercise regularly would be consistent with stoicism, if other stoical behaviours were demonstrated.

In summary, stoicism is perhaps too broad a concept to explain why fewer pain reports are made. It may be helpful to determine, however, whether a suspected stoic reports honestly, whether there is evidence of changes in threshold or tolerance on laboratory pain tests, and whether less expressive reporting represents a general coping style.

CONCLUSIONS

Beecher[35] described the Second World War soliders at a field hospital who, while aware of gunshot and other wounds, reported less pain and requested fewer drugs than civilians suffering similar wounds in surgical procedures. Similar apparent lapses of pain awareness appear at accident scenes and during athletic contests. Regular athletes also seem more stoical than others. Each of these situations represents a form of stress, and has led to research on the relationship between stress and pain perception. Much of the human research has focused on the effects of exercise, since it involves little risk and offers good experimental control. This research has documented the effects of acute exercise on pain responsivity and characterized the pain responses of people who exercise regularly.

Pain is reported to be lessened during and after exercise, but tolerance is unaffected. There is some evidence to suggest that this effect is mediated by an opioid anti-nociceptive system. Since the hypalgesic effect of exercise appears to be limited to threshold intensities of pain, however, this mechanism alone does not seem sufficient to explain inattention to gunshot wounds or bone fractures.

Laboratory studies may underestimate the magnitude of anti-nociception which is possible during life events. This is

because laboratory pain tests focus attention on the noxious stimulus, while atheltic competition and car accidents, for example, distract the subject from their injury. The ability of distraction to raise pain thresholds is well documented[36,37]. Thus, anti-nociception following exercise may be minimized in the laboratory, since this setting allows for little distraction. Another factor, stress, often coexists with distraction, and is little increased by exercise alone. Life events (e.g. battle) which involve exercise as well as more intense emotional arousal might be expected to produce more robust effects. For example, synergistic effects among three stressors have been demonstrated when the end points were autonomic variables, but not pain. Thus, greater levels of stress may activate endogenous anti-nociceptive systems more powerfully and further reduce the report of pain.

Although limited to three studies, available data also indicate that those who exercise regularly show greater pain tolerance than others (but similar thresholds), even without recent exercise. Greater tolerance is consistent with the reputed perseverance or stoicism of recreational athletes, and may account for episodes in which runners have carried on despite stress fractures and heart attacks. Further, when greater tolerance is coupled with elevation of the pain threshold as a result of acute exercise, these two factors may account for apparent instances of pain insensibility during athletic competition.

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